Abstracts

Oral 2

Occupational cancer I

O2.1 ESTIMATING THE NUMBER OF LUNG CANCER DEATHS IN GREAT BRITAIN DUE TO ASBESTOS EXPOSURE

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Introduction: Inhalation of asbestos fibres is known to cause two main kinds of cancer—mesothelioma and lung cancer. Although the vast majority of mesothelioma cases are generally accepted as being caused by asbestos, the number of asbestos related lung cancers cannot be determined directly because cases are not clinically distinguishable from those from other causes such as tobacco smoke. The aim of this study was to estimate the number of asbestos related lung cancers by modelling the relative lung cancer mortality among occupations within Great Britain in terms of smoking habits, mesothelioma mortality (as an index of asbestos exposure), and occupation type (as a proxy for socioeconomic factors).
Methods: Proportional mortality ratios for lung cancer and mesothelioma for the 20 year period 1980–2000 (excluding 1981) were calculated for occupational groups and smoking indicators were developed from three General Household Surveys carried out during the 1980s and 1990s. Poisson regression models were fitted and an estimate of the number of asbestos related lung cancers was produced by using the final model to estimate the number of lung cancer deaths in each occupation with no asbestos exposure and subtracting this from the predicted number of lung cancer deaths.
Results: The effect of asbestos exposure was weak in comparison to smoking habits and occupation type. The proportion of current smokers in occupational groups and average age started smoking were particularly important factors. Our estimate of the number of asbestos related lung cancers was between 2/3 and one death for every mesothelioma: equivalent to between 11 500 to 16 500 deaths during the period.
Conclusions: Asbestos related lung cancer is likely to have accounted for 2–3% of all lung cancer deaths among males in Great Britain over the last two decades of the twentieth century. Asbestos related lung cancers are likely to remain an important component of the total number of lung cancer deaths in the future as part of the legacy of past asbestos exposures in occupational settings.

O2.2 EXPLORING CANCER RISKS AMONG WORKERS IN THE SEMICONDUCTOR INDUSTRY IN TAIWAN

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Introduction: A retrospective cohort mortality study was conducted to explore cancer risks among workers in the semiconductor industry of Taiwan.
Methods: We obtained the employment information of workers in eight semiconductor companies from the Bureau of Labour Insurance, Taiwan. A total of 19 816 male and 27 610 female workers had been employed in these companies during 1980–2000. We identified workers with cancers by linking with the National Mortality Registry from the Department of Health, Taiwan. The NIOSH life table analysis system was used to generate person-years at risk and the expected numbers of death and to calculate standardised mortality ratios and their 95% confidence intervals for causes of death by using the referent rates from Taiwan.
Results: There was an apparently healthy worker effect for male workers: all deaths = 98; standardised mortality ratio (SMR) = 0.27, 95% CI 0.22 to 0.33 and all cancers = 27; SMR = 0.41, 95% CI 0.27 to 0.60). We found seven male leukaemia with SMR = 2.18 and 95% CI 0.87 to 4.49. In the analysis of a five year lag period there were five male leukaemia with SMR = 3.33, 95% CI 1.08 to 7.77. However, six of them had less than five years of employment. A healthy worker effect also existed in female workers: all deaths = 93; SMR = 0.63, 95% CI 0.51 to 0.77 and all cancers = 23; SMR = 0.68, 95% CI 0.42 to 1.02).
Conclusion: There seemed to be no apparent excess of cancer risk in the semiconductor industry so far. However, a healthy worker effect needs to be considered and leukaemia deserves future follow up studies.

O2.3 OCCUPATIONAL CHEMICAL EXPOSURE AND NON-HODGKIN'S LYMPHOMA

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Introduction: We assessed the association between non-Hodgkin's lymphoma (NHL) and occupational exposures to pesticides, solvents, metals, organic dusts, and PCBs in a case control study.
Methods: 694 cases of incident NHL during 2000 and 2001 and 694 community controls participated. A detailed occupational history was taken from each subject. For jobs with likely exposure to the chemicals of interest, additional questions were asked by telephone using modified job specific modules. An expert allocated exposures using the information in the job histories and the interviews. Odds ratios (OR) were calculated for each exposure adjusting for age, sex, region, and ethnic origin.
Results: Substantial exposure to any pesticide was associated with a trebling of risk of NHL (OR = 3.09, 95% CI 1.42 to 6.70). Subjects with substantial exposure to organochlorines, organophosphates, and ''other pesticides'' (all other pesticides excluding herbicides) and herbicides other than phenoxy herbicides had similarly increased risks of NHL, although the increase was statistically significant only for ''other pesticides''. The risk of NHL was increased by about 30% for exposure to any solvent with a dose response relation, subgroup analysis showed the finding was restricted to solvents other than benzene. Exposure to wood dust also increased the risk of NHL slightly. Exposures to other organic dusts, metals, and PCBs were not strongly related to NHL.
Conclusions: The risk of NHL appears to be increased by exposure to solvents other than benzene and possibly to wood dust. The increases in risk of NHL with substantial occupational pesticide exposure are consistent with previous work in this area.

O2.4 NON-HODGKIN'S LYMPHOMA AND DERMAL EXPOSURE TO PENTA- AND TETRACHLOROPHENOLS IN SAWMILLS

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Introduction: In a previous analysis of the British Columbia Sawmill Cohort we observed a weak association between non-Hodgkin's lymphoma (NHL) and exposure to fungicides containing chlorophenols. Several other studies have found chlorophenol exposed workers to have an increased risk of NHL, but the evidence has not been consistent. Animal studies have found pentachlorophenol (PCP) to be a more potent carcinogen than tetrachlorophenol (TCP). We have just completed an extended follow up of the BC Sawmill Cohort and assessed exposure to PCP and TCP separately.
Methods: The cohort consisted of 27 464 male workers employed for one year or more by 14 BC sawmills. Fatal (1950–95) and incident (1969–95) cancers were identified using national registries. Quantitative estimates of dermal exposure to PCP and TCP were developed using panels of experienced workers validated against urinary levels and company records for fungicide formulations. Dose response relations were assessed using Poisson regression.
Results: Overall, 49 NHL deaths (SMR = 1.02) and 92 incident cancers (SIR = 0.93) were observed. Both NHL mortality and incidence were associated with increasing PCP, but not with TCP exposure. For cumulative PCP exposure groups of 1–1.9, 2–4.9, and >5 full time equivalent years of dermal exposure (reference: <1), the relative risks

were 1.2, 2.4, and 1.8 (trend: p=0.06). The equivalent the relative risks in the incidence analysis were somewhat lower, 1.3, 1.9, and 1.7 (trend: p=0.02). The relative risks were much stronger with a 20 year lag; 3.0 (95% CI 1.2 to 7.3), 3.4 (95% CI 1.5 to 7.1), and 2.6 (95% CI 0.9 to 7.3) (trend: p=0.02) for mortality and 1.8 (95% CI 0.9 to 3.5), 2.1 (95% CI 1.1 to 3.7), 2.0 (95% CI 1.0 to 4.1) (trend: p=0.02) for incidence.
Conclusions: An association between NHL and pentachlorophenol exposure are consistent with other studies. It is not clear why the relative risks were higher for mortality than incidence. The lack of a monotonically increasing dose response may be the result of misclassification or small numbers.

O2.5 COLON CANCER, SERUM OMEGA-3 FATTY ACIDS, AND AGRICULTURAL WORK: A NESTED CASE CONTROL STUDY IN A NATIONAL COHORT OF FARMERS

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Introduction: Colon cancer accounts for about 10% of cancers in developed countries. Farmers generally show a reduced risk of colon cancer. Less sedentary work and different diet habits in farmers compared with the general population may possibly explain the lower colon cancer incidence in farmers. The aim of this study was to assess the associations of prediagnostic serum fatty acids, information on farm work, and physical activity according to censuses and health survey investigations, and cancer of the colon.
Methods: 177 colon cancer patients reported to the Cancer Registry in 1976–2000 who were included in an earlier established national cohort of farmers' families and who also contributed serum to the Janus Serum Bank in 1974–91 were selected as cases for the study. Controls were sampled from the same study base, using matching 1:1 by age, sex, calendar year, and county. Data on farm production according to agricultural censuses 1969–89 and health survey information collected at the time of the blood sampling were obtained from Statistics Norway and the Norwegian Institute of Public Health, respectively. Prediagnostic levels of phospolipid fatty acids were determined in the serum samples.
Results: Colon cancer was inversely associated with the Ω-3 fatty acid EPA (eicosapentaenoic, 20:5), OR=0.43 (95% CI 0.24 to 0.76 in the highest quartile compared to the lowest, adjusted for age and sex). Colon cancer was associated with farm acreage (OR 1.66, 95% CI 1.07 to 2.54), but not with indices of yearly farm work input or self-evaluated work intensity or with the body mass index. There was also a weak negative association with the Ω-3 fatty acid DHA (docosahexaenoic, 22:6).
Conclusion: Colon cancer may be associated with low serum Ω-3 fatty acids. A low dietary intake of these essential fatty acids thus may be a risk factor for colon cancer.